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Attempted Nonsurgical Electrical Ablation of Accessory Pathways Via the Coronary Sinus in the Wolff-Parkinson-White Syndrome

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Previous canine experiments suggested that transvenous catheters placed in the coronary sinus could be used to deliver limited energy shocks, resulting in fibrosis in the atrial wall and coronary sulcus with sparing of the coronary artery. From the distribution of the fibrosis, it appeared that this approach could be used for attempted ablation of accessory pathways in patients with the Wolff-Parkinson-White syndrome.

Eight patients with symptomatic Wolff-Parksinson-White syndrome underwent electrophysiologic testing with attempted ablation of 10 accessory pathways. Shocks were limited to 40 to 80 J, except in one patient who received shocks of 100 and 150 J. From 2 to 26 shocks were given to each accessory pathway. All the accessory pathways were blocked completely immediately after the shocks. Subsequently, evidence of accessory pathway conduction recurred in each patient. Three had early promise of long-term improvement after the procedure, with prolongation of the refractory periods of the accessory pathways during the remainder of the initial hospitalization. Several weeks later, however, there was evidence of return toward original values in two of these. Another patient who appeared not to benefit during her initial hospitalization returned 7 weeks later with very depressed accessory pathway conduction, possibly due to developing fibrosis. The only significant complication occurred in the patient receiving shocks of 100 and 150 J; he had apparent rupture of the coronary sinus requiring pericardial drainage. In two patients in whom nonsurgical ablation was not successful, intraoperative mapping showed that the accessory pathway was located in an area of fibrosis at the site of the attempted ablation.

In summary, nonsurgical electrical ablation of accessory pathways via the coronary sinus may be successful using limited energy levels in a few patients. The procedure remains experimental, and widespread application must await more effective means of delivering the shocks.

There has been a recent surge of interest in internal cardiac stimulation using energy sufficient for cardioversion, defibrillation and localized ablation of cardiac tissue (1–14). Ablation of the atrioventricular (AV) conduction system using shocks delivered through conventional or modified multipolar electrode catheters appears to be a safe and effective technique for selected patients with drug-resistant supraventricular tachycardia (5,7–8,13). The technique has been applied successfully to ventricular tachycardia (9), and septal accessory (Kent) pathways have been ablated (5,10,12,14).

In patients with the Wolff-Parkinson-White syndrome, arrhythmias relevant to the accessory pathway include rapid anterograde conduction of atrial fibrillation, and AV reciprocating tachycardia. In the latter, the accessory pathway is usually used for retrograde conduction, and the normal pathway for anterograde conduction. The rationale for treatment in both instances is to block or increase the refractoriness in the accessory pathway, thereby preventing or slowing the conducted arrhythmia.

In patients with the Wolff-Parkinson-White syndrome, the accessory pathway may lie in close proximity to the coronary sinus (15,16). Extensive canine experiments in our laboratory (17) demonstrated that limited energy levels delivered to the coronary sinus could result in histopathologic changes likely to involve the accessory pathway, but sparing

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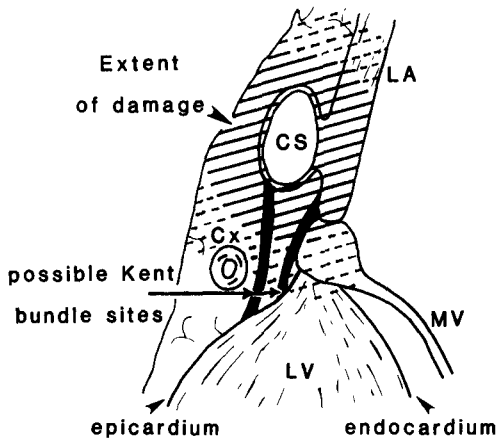

Figure 1. Schematic representation of the effects of attempted electrical ablation of accessory pathways by way of the coronary sinus. Cross section of the left posterior heart, showing the left atrium (LA) (**above**), with the endocardium to the **right** and the epicardium to the **left**; the left ventricle (LV) (**below**), with the mitral valve (MV) anulus, circumflex coronary artery (Cx) and coronary sinus (CS) in the **middle**. The **heavy lines** represent the locations of possible accessory pathways. Coronary sinus shocks in canine experiments (17) resulted in damage and fibrosis to the **hatched area** with sparing of the coronary artery.

the coronary artery (Fig. 1). This technique has now been applied to symptomatic patients with the Wolff-Parkinson-White syndrome with left-sided accessory pathways approached via the coronary sinus. This paper is not intended to report in detail the conventional management data of the patients involved, but rather to address the question of whether accessory pathways in proximity to the coronary sinus can be ablated safely using the techniques described.

Methods

Patients. Eight patients were studied after giving informed consent to a protocol approved by the Institutional Review Board at Montefiore Hospital. The highly experimental nature of the procedure was carefully explained, along with the alternatives of conventional and experimental medications or surgical ablation for those with drug-resistant or life-threatening arrhythmias. Their ages ranged from 18 to 54 years. All had either overt Wolff-Parkinson-White syndrome with symptomatic tachycardia related to accessory pathway conduction, or tachycardia due to retrogradely conducting accessory pathways (Table 1). All had failed to respond to one to six (mean three) antiarrhythmic drugs. Patients 1, 4, 5 and 7 had syncope; Patients 2, 6 and 8 had near syncope. Patient 2 had ventricular rates approaching 300 beats/min during atrial fibrillation with lightheadedness and chest pain. Patient 3 had 20 to 40 episodes of tachycardia per day in spite of multiple drug trials. Patient 5 had severe mitral stenosis, recurrent after a previous valvotomy. A variety of medications had failed to control her episodes of

Table 1. Clinical Summary of Eight Patients Before and After Ablation

Patient no. (age [yr]/sex) Clinical Arrhythmia	Patient 1 (19/ F) AF		Patient 2 (41/M) AF, AVT		Patient 3 (23/M) AVT		Patient 4 (26/F) AVT	
	Before	After	Before	After	Before	After	Before	After
Frequency of episodes								
Without drugs	1 to 2/mo	0	0 to 5/mo	0	20 to 40/day	8 to 20/day	1/mo	(0)†
With drugs*	1 to 2/mo	0	0 to 5/mo	0	20 to 40/day	0 to 1/day	1/mo	0
Severity of episodes								
Without drugs								
Palpitation	+	0	+	(0)	+	+	+	(0)†
Syncope	+	0	Near	(0)	0	0	+	(0)†
With drugs								
Palpitation	+	0	+	0	+	+	+	+
Syncope	+	0	0	0	0	0	+	0
Drug treatment								
Ineffective	DPH§	–	P	Q,D	N,PA,P	V,PA	D,Q,D+Q,P	PA
Effective	—	N, NM	–	N,PA	–	V+D	–	PA+V,N
Follow-up (mo)		25		24		21		19
Associated conditions		0		0		0		0

*Most effective regimen. †No episodes during 1 week off drugs before latest follow-up. ‡Severe congestive heart failure was the most serious problem; patient underwent surgery 8 weeks after attempted ablation (see text). §Patient was treated with phenytoin for several years because of syncopal spells attributed to epilepsy; full neurologic work-up negative. ¶Aprindine discontinued because of severe hepatotoxicity. AF = atrial fibrillation with anterograde conduction over accessory pathway; After = after ablation; AP = aprindine; AVT = atrioventricular tachycardia (the typical reciprocating tachycardia of the Wolff-Parkinson-White syndrome); Before = before ablation; CAD = coronary artery disease (Patient 6 postinfarction, not a surgical candidate; Patient 7 has a proximal left anterior descending coronary artery obstruction, angioplasty done or possible surgery for both Wolff-Parkinson-White

atrial fibrillation, conducted at rates up to 200 beats/min and causing profound hemodynamic decompensation. Although mitral valve replacement was planned, nonsurgical ablation was attempted so that, if successful, operative time would be reduced.

Cardiac catheterization. This procedure with coronary arteriography was recommended in all patients, and mandatory after the third patient, primarily to assess the relation between the coronary sinus and the left circumflex coronary artery. Although canine data suggested a minimal risk of coronary artery damage from coronary sinus shocks (17), it was decided not to attempt an ablation where the two vessels were in proximity. Three additional potential candidates were excluded because the accessory pathway was adjacent to a large circumflex artery.

Exercise testing. This test, using a bicycle ergometer, was performed before electrophysiologic studies and at intervals thereafter. The shortest ventricular cycle length resulting from accessory pathway conduction was recorded as the anterograde accessory pathway conduction limit.

Electrophysiologic studies. These were carried out in a conventional manner, with percutaneous insertion of multipolar catheters under local anesthesia. The effective and functional refractory periods of the normal and accessory conduction pathways were assessed, when possible, using atrial and ventricular programmed stimulation during sinus rhythm and pacing. The shortest pre-excited ventricular cycle lengths (conduction limits) were recorded during atrial fibrillation when observed. Anterograde accessory pathway conduction limits were determined in all patients during incremental ramp atrial pacing (18). The shortest pre-excited atrial cycle length during incremental ramp ventricular pacing was recorded as the retrograde accessory pathway conduction limit. Incremental ramp pacing employed a continuously increasing pacing rate, increasing from 80 beats/min in increments of approximately 2 beats/min every second. In some instances, the refractory periods and conduction limits could not be measured because of the induction of fibrillation or tachycardia, or during exercise because accessory pathway conduction persisted up to the maximal heart rate achieved. In these instances, a "less than" symbol (<) was recorded. Atrial stimulation was performed at several sites in the right atrium and in the left atrium via the coronary sinus. All stimuli were delivered using a Devices/Digitimer 4279 modular pacing system, with outputs at four times diastolic threshold and 1 ms pulse duration.

Electrophysiologic studies in these patients were continued over several days using an electrode catheter that remained in the coronary sinus. Therefore, the anterograde conduction intervals and refractory periods reported in the study are based on coronary sinus stimulation. In Patients 3, 4 and 8, a second catheter lead was left in the right ventricle for determination of retrograde refractory periods and conduction limits.

Intracardiac bipolar mapping was performed to determine activation sequence during pacing, and during regular tachycardia if induced. Atrial mapping was performed during retrograde conduction using catheters situated at the high

Table 1. *(continued)*

Patient no. (age [yr]/sex) Clinical Arrhythmia	Patient 5 (37/F) AF		Patient 6 (54/M) AVT		Patient 7 (36/F) AF		Patient 8 (18/F) AVT	
	Before	After	Before	After	Before	After	Before	After
Frequency of episodes								
Without drugs	2 to 3/mo	—	Daily	Daily	(1)	(0)	4/mo	(0)
With drugs*	0 to 3/mo	(0)	8 to 40/mo	0 to 2/mo	(0)	(0)	4/mo	(0)
Severity of episodes								
Without drugs								
Palpitation	+‡	(0)	+	+	+	(0)	+	(0)
Syncope	+‡	(0)	Near	0	+	(0)	Near	(0)
With drugs								
Palpitation	+‡	(0)	+	0	(0)	(0)	+	(0)
Syncope	+‡	(0)	Near	0	(0)	(0)	0	(0)
Drug treatment								
Ineffective	D,P,PA,N,Q	—	D,P,PA,V	D,P,PA,V	PA,Q	—	PA,P	—
Effective	AP¶	—	—	D+PA+V	—	(PA)	—	PA
Follow-up (mo)		15		6		4		4
Associated conditions		RHD		CAD		CAD		0

syndrome and coronary artery disease if both nonsurgical treatments fail); D = digoxin; DPH = diphenylhydantoin (phenytoin); F = female; M = male, mo = month; N = disopyramide; NM = no medications needed; P = propranolol; PA = procainamide; Q = quinidine; RHD = rheumatic heart disease with severe mitral stenosis (Patient 5 underwent surgical treatment for both mitral stenosis and Wolff-Parkinson-White syndrome [see text]); V = verapamil; + = present; 0 = absent; (0) = limited data or brief trials only. Parentheses after patient number indicate the patient's age (in years) and gender.

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right atrium, His bundle area and multiple sites around the tricuspid anulus using a Gallagher (modified Brockenbrough) catheter (15); left atrial mapping was performed using a catheter advanced as far as possible into the coronary sinus and withdrawn in a stepwise fashion during each tachycardia or pacing condition. In Patients 5 to 8, a hexapolar or octapolar catheter with 8 to 10 mm interelectrode spacing (Cordis, USCI) permitted mapping in the coronary sinus without moving the catheter. The site of earliest anterograde ventricular activation was determined during sinus rhythm and atrial pacing using catheters in the same sites as used for atrial mapping. Mapping data were interpreted from the interval from the pacer stimulus or onset of atrial or ventricular depolarization to the peak local deflection in the ventricle or atrium, respectively, for anterograde and retrograde conduction.

Ablation of the accessory pathway. This procedure was attempted through the electrodes at the estimated site of the accessory pathway recording the shortest conduction interval. Additional shocks to distal or proximal sites, or both, were delivered in Patients 2 to 5 and 7 and 8 (see Results). The patients were anesthetized with methohexital (Brevital) and paralyzed by succinylcholine to prevent laceration of the coronary sinus due to skeletal muscle stimulation. Bipolar shocks were used in Patients 1 to 4 and 7 and 8 and unipolar shocks were used in Patients 5 and 6. Unipolar shocks were cathodal, with anodal plates on the upper anterior chest and abdomen. In Patients 1 and 2, asynchronous shocks were provided by an Electrodyne C-100M defibrillator. Synchronous shocks were delivered in subsequent patients by a Physio-Control Lifepak 911. Multichannel surface and intracardiac recordings were made continuously during the procedure. The number and energy of the shocks varied depending on effects and with increasing experience as discussed in the Results section.

Other procedures and methods. Twelve lead electrocardiograms included multiple baseline recordings before the procedure, intermittent records during the electrophysiologic study, every 12 hours the next day and daily recordings thereafter during the hospitalization. Twenty-four hour electrocardiographic (Holter) recordings were made in each patient, both before and after the attempted ablation procedure. Blood was drawn for cardiac enzyme levels two or more times before the ablation attempt and afterwards at 2 hours and then every 12 hours for 3 days

Attempted ablation procedures were performed in the presence of a cardiothoracic surgeon and with the help of senior members of the Anesthesiology Department. An operating room was available; the first two patients were observed in the recovery room for several hours after the procedure to facilitate rapid transport to the operating room in the event of hemodynamic embarrassment suggestive of rupture. Thereafter, the patients were transferred initially to the coronary care unit and subsequently to the intermediate care unit and general medical wards.

Follow-up. The refractory periods or conduction limits, or both, of normal and accessory pathways were determined both immediately after the ablation attempt and during the succeeding days. Patients 1 to 5 and 8 were evaluated 6 to 9 weeks after the procedure with a careful physical examination, electrocardiogram and exercise test, all in the absence of antiarrhythmic medications. Patients 4, 5 and 8 underwent a repeat electrophysiologic study without attempted ablation 9, 8 and 6 weeks, respectively, after the initial study.

Results

An overall clinical summary is provided in Table 1.

Baseline electrocardiographic and exercise data. In Patients 1, 2, 4, 5 and 8, delta waves were present continuously in each of multiple electrocardiograms recorded before the procedure, including the 24 hour tapes. Delta waves were occasionally absent in Patient 7 and were visible in Patient 3 primarily during sinus rates below 75 beats/min. Baseline exercise tests were, therefore, not performed in Patient 3; in addition, Patients 5 and 6 were too ill to undergo baseline exercise testing. Delta waves persisted throughout the exercise tests in Patients 1, 2, 4 and 8, who exercised to maximal rates of 188, 175, 175 and 180 beats/min, respectively. An exercise test in Patient 7 showed delta waves throughout to a rate of 177 beats/min, at which point she developed an irregular wide complex tachycardia at 300 beats/min; exercise testing was not repeated after the attempted ablation pending coronary revascularization.

Electrophysiologic data before attempted ablation (Table 2). Patient 3 had poor anterograde accessory pathway conduction with all refractory periods in excess of 600 ms. Delta waves were never observed in Patient 6. The anterograde functional refractory periods for Patients 1, 2, 5, 7 and 8 ranged between 250 and 300 ms; for Patient 4, the functional refractory period could not be measured because of tachycardia induction. The shortest cycle length during atrial fibrillation was shorter, ranging from 160 to 290 ms in Patients 2, 4, 5 and 8. The ramp conduction limit was generally similar to the extrastimulus functional refractory period, except in Patient 2, in whom it was more comparable with the conduction limit observed during atrial fibrillation.

Retrograde conduction through the accessory pathway was present in Patients 2, 3, 4, 6 and 8 and absent in Patients 1 and 5. Patient 7 had retrograde conduction only through a lateral pathway with a long refractory period that was not subjected to a shock. Retrograde ramp conduction limits tended to be shorter than the functional refractory period.

Left-sided accessory pathways were identified by retrograde or anterograde mapping in each of these patients (Fig. 2). In Patients 3, 4 and 7, there was evidence of two left-sided accessory pathways.

Table 2. Refractory Periods and Conduction Limits of Accessory Pathways

	Anterograde					Retrograde		
	Refractory Periods (ms)		Conduction Limits (minimal RR) (ms)			Refractory Periods (ms)		Conduction Limits (minimal AA) (ms)
	ERP*	FRP	AF	Ramp	Exercise	ERP	FRP	Ramp
Patient 1								
Baseline	260	280	—	290	<320	Block	Block	Block
After shock								
Immediately	Block	Block	—	Block	—	Block	Block	Block
0.5 to 1.0 h	—	—	—	330	—	Block	Block	Block
7 wk	—	—	—	—	400 to 500	—	—	—
7 mo	—	—	—	—	330 to 500	—	—	—
13 mo	—	—	—	—	<330	—	—	—
Patient 2†								
Baseline	240	280	200	215	<340	220	300	260
After shock								
Immediately	Block	Block	—	Block	—	Block	Block	Block
0.5 to 1.0 h	—	—	—	<250	—	—	—	260
4 days	—	—	230	275	<400	—	—	—
6 wk	—	—	—	—	<340	—	—	—
Patient 3								
Baseline	>600	>600	—	>600	—	340	350	310
After shock‡								
Immediately	>600	>600	—	>600	—	Block	Block	Block
0.5 to 1.0 h	>600	>600	—	>600	—	Block	Block	Block
10 days	>600	>600	—	>600	>650	270	300	290
Patient 4†								
Baseline	<300	<300	160	<300	<340	<270	<280	—
After shock								
Immediately	Block	Block	—	Block	—	Block	Block	Block
0.5 to 1.0 h	—	—	—	270	—	Block	Block	Block
6 days	—	—	—	270	—	<280	<320	<280
9 wk	<250	<280	235	<280	<375	AF	AF	AF
Patient 5								
Baseline	290	300	290	320	—	Block	Block	Block
After shock								
Immediately	Block	Block	Block	Block	—	Block	Block	Block
0.5 to 1.0 h	410	410	—	410	—	Block	Block	Block
5 days	300	330	—	300	—	—	—	—
8 wk	260	320	—	290	—	—	—	—
Patient 6								
Baseline	Block	Block	Block	Block	Block	<220	<280	<240
After shock								
Immediately	—	—	—	—	—	Block	Block	Block
0.5 to 1.0 h	—	—	—	—	—	Block	Block	Block
Patient 7								
Baseline	270	290	—	290	<340	500§	520§	480§
After shock								
Immediately	Block	Block	—	Block	—	—	—	—
0.5 to 1.0 h	310	310	—	330	—	—	—	—
7 days	310	310	—	295	—	—	—	—
Patient 8								
Baseline	200	250	230	260	<330	230	270	270
After shock								
Immediately	Block	Block	Block	Block	—	Block	Block	Block
0.5 to 1.0 h	220	310	—	310	—	—	—	—
5 days	270	285	—	300	<370	280	305	275
6 wk	240	275	—	230	—	200	200	240

*Effective and functional refractory periods (ERP and FRP, respectively) during pacing at cycle length 500 to 600 ms, except Patient 4 at 9 weeks (430 ms), Patient 5 (410 to 440 ms) and Patient 8 (400 ms); all were in an unmedicated state, except Patient 5 who required propranolol to prevent incessant atrial fibrillation. †In Patients 2 and 4, refractory periods were difficult to assess because of exquisite ease of tachycardia and induction of atrial fibrillation. ‡In Patient 3, intermittent delta waves were observed during sinus rhythm in the baseline state, but never after attempted ablation. §Retrograde conduction over a distal accessory pathway, remote from that conducting anterogradely; because of a long refractory period, no shocks were directed at this distal pathway. — = no data available; AF = atrial fibrillation (numbers are shortest consecutive preexcited RR interval during atrial fibrillation [see definitions of conduction limits]; Conduction limits: anterograde conduction limit = shortest consecutive preexcited RR interval (ventricular cycle length) during atrial fibrillation, incremental atrial ramp pacing and exercise; retrograde conduction limit = shortest consecutive preexcited AA interval during ventricular pacing.

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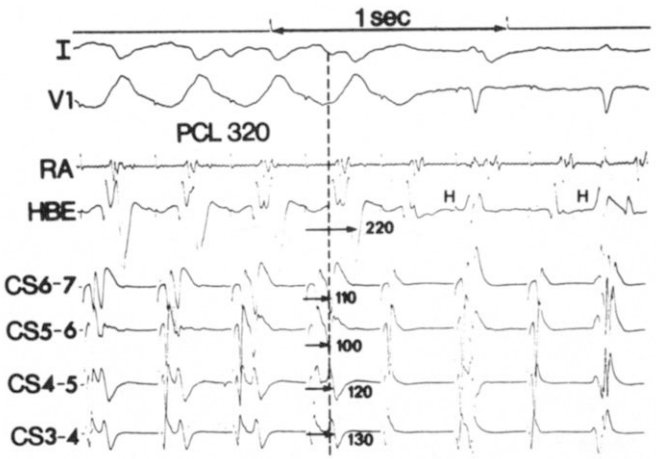

Figure 2. Patient 5. Mapping and anterograde ramp conduction limits of the left-sided accessory pathway. Peripheral leads I and V_1 are displayed, together with recordings from the high right atrium (RA), His bundle area (HBE) and multiple bipolar recordings from an octapolar catheter with electrodes 8 mm apart, all located in the coronary sinus (CS) with leads 1 and 2 (not shown) most distal. During incremental ramp pacing at a cycle length (PCL) of 320 ms, accessory pathway conduction ceases, with normalization of the QRS pattern. This represents the anterograde ramp conduction limit of the accessory pathway. The **vertical dashed line** shows the earliest local ventricular activation during anomalous conduction recorded on coronary sinus leads 5 and 6, 100 ms after the pacer stimulus in coronary sinus leads 1 and 2. The time from the stimulus to local ventricular activation is indicated (in ms) by the **horizontal arrows**.

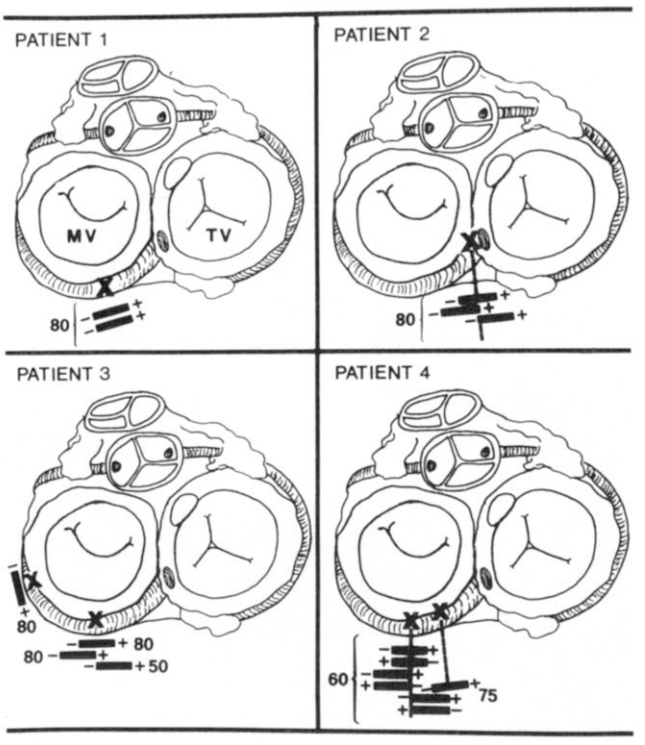

Figure 4. Schematic location, energy and polarity of bipolar shocks used in four patients. The heart is sectioned at the plane between the atria and the ventricles, and is viewed from behind and above. The mitral valve (MV) and tricuspid valve (TV) are illustrated with the ostium of the coronary sinus visible in the **lower left corner** of the tricuspid valve ring leading into the coronary sinus which lies posterior to (**below**) the mitral valve. Site X of accessory pathways is based on intracardiac mapping. (+) and (−) indicate polarity of bipolar shocks delivered in the coronary sinus and the **numbers** indicate the joules delivered. The **heavy lines** indicate the position and sequence (**top to bottom**) of the bipolar shocks.

Attempted ablation of accessory pathways. After achievement of satisfactory anesthesia, shocks were delivered to catheter electrodes placed at the estimated site of the accessory pathway. In all of these patients, the accessory pathways lost the ability to conduct immediately after the shock (Table 2, Fig. 3). Variations in technique resulted in three groups.

Group 1: bipolar shocks tightly centered on the accessory pathway site (Patients 1 to 4) (Fig. 4). One to six bipolar shocks with energies ranging between 60 and 80 J (delivered) were used on each pathway identified in Patients 1 to 4. In Patients 2, 3 and 4, the catheter lead position was moved 0.5 to 1 cm between shocks to create a modest overlap at three accessory pathway sites (Fig. 4). In Patient 4, the polarity of shocks was alternated. In Patient 3, a more lateral pathway could not be "bracketed"; that is, the ear-

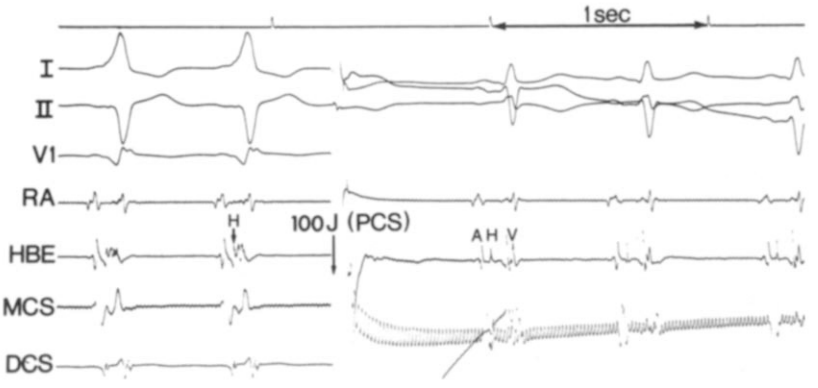

Figure 3. Patient 2. Normalization of the electrocardiogram during attempted ablation of the accessory pathway. The electrodes at the proximal coronary sinus (PCS) are connected to the defibrillator rather than the recording device, and a shock of 80 J (100 stored) is delivered asynchronously during sinus rhythm, with normalization of conduction and prolongation of the HV interval to normal. Peripheral leads I, II and V_1 are displayed, together with recordings from the His bundle region (HBE), mid coronary sinus (MCS) and distal coronary sinus (DCS).

liest depolarization was recorded from the most distal position to which the catheter could be advanced (Fig. 5).

Group 2: unipolar shocks (Patients 5 and 6). Patient 5 received a total of 26 unipolar shocks of 40 to 50 J: 6 each through electrodes 4, 5, 6 and 7 of the octapolar lead, and 2 through electrode 8. The accessory pathway in this patient appeared to be nearest to electrodes 5 and 6 (Fig. 2). It was, therefore, intended to distribute the shocks in an area centered around the estimated site of the accessory pathway. For half of these shocks, the anode was a large adhesive plate on the upper anterior chest; for the other half, a similar plate was positioned on the upper anterior abdomen. In this patient, there was also immediate normalization of the QRS complex.

Patient 6 received two unipolar shocks of 150 and 100 J, respectively, centered at the accessory pathway site. Block immediately occurred in this pathway located at the junction of the proximal and middle thirds of the coronary sinus. However, the patient developed cardiac tamponade and required immediate pericardial drainage, which restored him to a stable condition.

Group 3: bipolar shocks distributed around the accessory pathway site (Patients 7 and 8). Five shocks of 75 J were administered in Patient 7. These were distributed over 3 cm centered around her accessory pathway located at the junction of the proximal and middle thirds of the coronary sinus and resulted in immediate block in the pathway.

The accessory pathway in Patient 8 was located in the region of the proximal coronary sinus. Five shocks of 50 J were distributed over a 2 cm range and produced block in the accessory pathway. When delta waves reappeared after 15 minutes, four additional unipolar shocks of 300 J were delivered in the right atrium to posterior septal sites in proximity to the ostium of the coronary sinus, and again produced transient block.

Electrophysiologic data after attempted ablation of accessory pathway (Table 2). Incremental ramp pacing was carried out periodically during the hour after attempted ablation and on succeeding days to estimate conduction limits. Refractory period determinations using the extrastimulus technique were also used during this period. As noted previously and outlined in Table 2, all accessory pathways subjected to attempted ablation were blocked in each patient immediately after the procedure. During the next hour, there was a gradual return of accessory pathway conduction in six patients, although refractory periods were often markedly prolonged compared with baseline values (Fig. 6). Assessment of refractory periods or conduction limits, or both, was carried out again 4 to 10 days after the attempted ablation procedure in six patients (Patients 2 to 5, 7 and 8), by which time only Patients 2 and 8 showed persistence of prolonged refractory periods or conduction limits. All of these patients were subsequently given antiarrhythmic drug therapy, with serial electrophysiologic

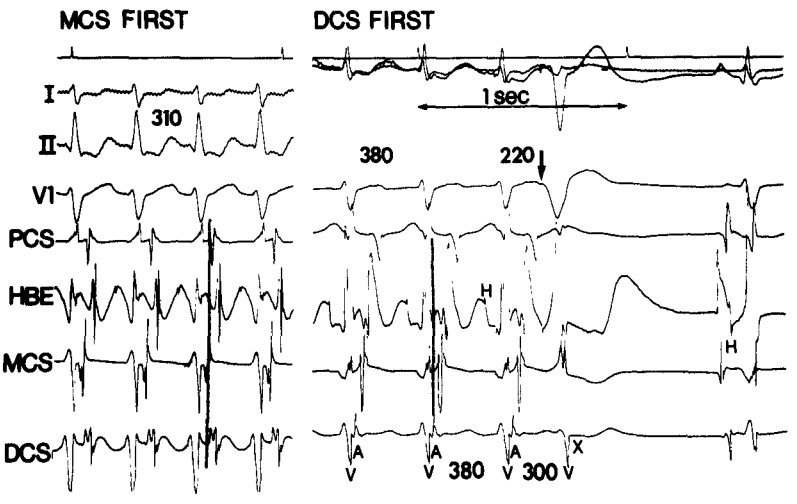

Figure 5. Patient 3. Summary of mapping showing evidence of double accessory pathways. Peripheral leads I, II and V_1 are shown, together with recordings from the proximal coronary sinus (PCS), His bundle area (HBE), mid coronary sinus (MCS) and distal coronary sinus (DCS). In these illustrations, ventricular activity (V) precedes atrial depolarization (A) during normodromic tachycardia. **Left panel**, Tachycardia at a cycle length of 310 ms. The **vertical dashed line** indicates that the first atrial depolarization occurs in the mid coronary sinus lead. **Right panel**, Tachycardia with initial atrial depolarization in the distal coronary sinus. The cycle length is longer at 380 ms because of slower AV nodal conduction rather than to a longer circuit length (see text). In both tachycardias, anterograde conduction is through the normal pathway, with normal QRS complexes each preceded by a His bundle potential (H). The tachycardia using the distal coronary sinus pathway is terminated by a ventricular extrastimulus (**arrow pointing down**) delivered 220 ms after the preceding ventricular event. Labeling on the distal coronary sinus lead indicates that the extrastimulus delivered to the right ventricle was conducted to the left ventricle with some delay, resulting in a VV interval of 300 ms as recorded from lead DCS; this was sufficient to terminate the tachycardia due to retrograde block (absence of the atrial depolarization indicated by the X).

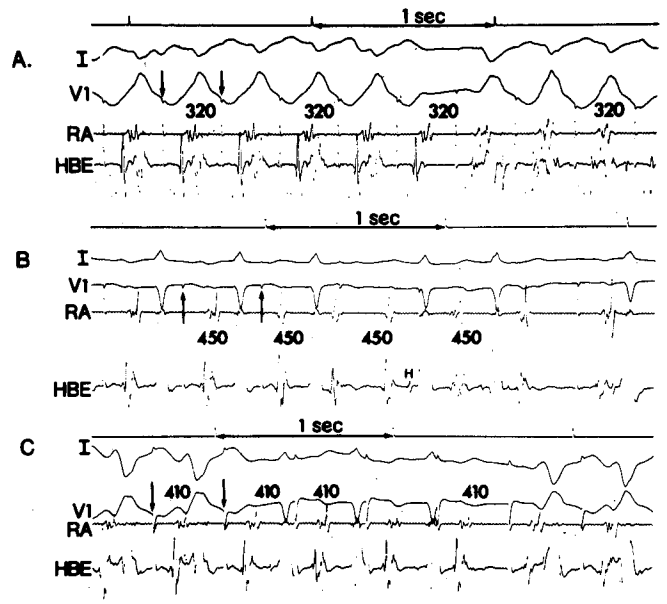

Figure 6. Patient 5. Sequential changes in accessory pathway conduction during the attempted ablation procedure. Peripheral leads I and V_1 are shown, together with recordings from the right atrium (RA) and His bundle areas (HBE). Modified ramp pacing was used, with the cycle length maintained for purposes of illustration. All pacing stimuli (**vertical arrows**) were delivered in the distal coronary sinus. **A**, Baseline recording before attempted ablation. During pacing at a cycle length of 320 ms, there is occasional failure of accessory pathway conduction. **B**, Immediately after attempted ablation, there is no evidence of accessory pathway conduction, with normalization of the QRS interval and Wenckebach conduction at the AV node level during pacing at a cycle length of 450 ms. **C**, One hour after attempted ablation, there has been some return of accessory pathway conduction. However, block in the accessory pathway occurs during pacing at a cycle length of 410 ms.

testing to determine the best regimen for prevention of tachycardia induction or prolongation of the accessory pathway refractory periods.

Exercise testing after attempted ablation procedure (Table 2). Five patients underwent exercise testing after attempted ablation while receiving no antiarrhythmic medications. In Patient 3, delta waves were seen only intermittently during slow sinus rhythm before ablation and not at all after the attempted ablation. In Patients 2, 4 and 8, delta waves persisted during exercise tests performed both several days and from 6 to 9 weeks (Patients 2 and 4) after attempted ablation. In Patient 1, whose delta waves had returned almost immediately after the attempted ablation procedure, the course was different. Before the procedure, her delta waves had persisted during multiple electrocardiograms, Holter recordings and exercise testing. She returned for exercise testing 7 weeks after the procedure, and in the absence of medication (confirmed by drug blood levels), delta waves became intermittent with exercise at rates greater than 100 beats/min and were gone entirely at rates above 120 beats/min. Repeat exercise testing at 7 and 13 months, however, showed a progressive increase in the rates associated with persistent delta waves during exercise.

Clinical course. The procedure was well tolerated by all except Patient 6, who required surgical intervention for cardiac tamponade. Patients 3 and 4 complained of pericardial-type pain for several days without objective findings. There were no other complications. Serial electrocardiograms were recorded for 3 days and were entirely normal in Patient 1, whose QRS complexes were normalized by disopyramide. There were no sequential electrocardiographic changes suggestive of myocardial injury. Cardiac enzymes, including creatine kinase-MB, were normal as recorded 2 hours after attempted ablation and then every 12 hours for 3 days except in Patient 8 who received 300 J septal shocks. All patients promptly returned to their usual state of health and physical activity, except Patient 6, whose recovery was slower.

Follow-up. At the time of this writing, Patient 1 has been free of symptoms for the 25 months after her procedure with medications discontinued after the first 4 weeks. Patients 2 to 4 were successfully treated with antiarrhythmic drugs (Table 1). Patient 3, one of those with double accessory pathways, continued to have frequent episodes of tachycardia while taking no medications. However, the typical rate of his episodes was reduced from 180 to 140 beats/min. These slower tachycardias may have been due to longer AV nodal conduction (Fig. 5). Consistently slower tachycardias could also be related to damage to his accessory pathways or successful ablation of his more medial pathway, with slower tachycardia due to a longer circuit involving the more lateral pathway.

Patient 5 underwent her scheduled operative procedure for mitral valve replacement 8 weeks after the attempted ablation procedure. On the day before operation, electrophysiologic testing revealed no evidence of refractory period prolongation in the accessory pathway (Table 2). Intraoperative mapping was performed to locate the Kent bundle for surgical division. It was noted that the entire atrium was large and diffusely fibrotic, appearing pearly gray in color rather than pink. Intraoperative mapping identified the same location for the Kent bundle as predicted from mapping in the catheterization laboratory. Careful inspection of the coronary sulcus contents at this point revealed considerable fibrosis and occlusion of the coronary sinus consistent with the findings in the animal studies (17) (Fig. 1). After the operation, there was no further evidence of accessory pathway conduction, either clinically or during electrophysiologic studies.

Patient 6 is being followed up and may require surgery; he is not tolerating medications well. Patient 7 underwent successful coronary angioplasty and is receiving medical therapy for arrhythmia prophylaxis. Patient 8 was readmitted after 6 weeks to assess the long-term effects of attempted

ablation. Refractory periods and conduction studies were similar to baseline values, and he underwent successful surgical division of his anomalous pathway. As in Patient 5, the pathway was located at the site predicted during the electrophysiologic testing, together with fibrosis resulting from the attempted ablation procedure.

Discussion

The possibility of a nonsurgical "cure" of the Wolff-Parkinson-White syndrome is attractive because it would free patients from life-long dependence on medications and require a shorter convalescence than would surgical approaches. The procedures described in this study were performed after a series of canine experiments (17) had demonstrated that limited energy bipolar shocks delivered to the coronary sulcus could result in desirable degrees of fibrosis without affecting the circumflex coronary artery.

Limited efficacy of the procedure. In this series, attempted nonsurgical ablation of left-sided accessory pathways resulted in complete but transient block of accessory pathways in each patient. Patient 1 has had a favorable long-term effect, being free of recurrent episodes without medications for 2 years. In Patient 2, the accessory pathway refractory periods may have been permanently prolonged. In Patient 3, recurrent tachycardias in the unmedicated state are slower and better tolerated, but this may be due to slower AV nodal conduction (Fig. 5), rather than to ablation of one of his two accessory pathways. Patients 4 to 8 have not derived apparent benefit from their attempted ablation procedures. There was one important complication in this group of patients (cardiac tamponade in Patient 6), apparently due to rupture of the coronary sinus and requiring surgical intervention.

Exercise studies performed before electrophysiologic studies accurately predicted refractory periods shorter than the shortest exercise cycle length (19). Very short refractory periods are better defined by electrophysiologic testing or isoproterenol infusion (20). Serial exercise testing in Patient 1 has shown progressive shortening of the long postablation accessory pathway conduction limit, although arrhythmias have not yet recurred.

Technical considerations. There may be several reasons for the limited efficacy of the procedure. Successful ablation of the normally located AV conduction system or sites of ventricular tachycardia has required shocks of several hundred joules (5–10). In all but one of our patients, shocks were limited to 80 J or less to minimize the possibility of rupturing the coronary sinus. During the canine studies in our laboratory (17), rupture of the coronary sinus with subsequent exsanguination occurred with energies in excess of 200 J. Apparent rupture of the coronary sinus occurred in Patient 6, the only patient to receive higher energy shocks of 100 and 150 J. Even at these energy levels, block of the accessory pathway was transient.

Ablation techniques performed within the cardiac chambers (5–14) have used unipolar shocks to "direct" the energy at the target tissues, using an anodal plate applied to the skin. The variability of possible Kent bundle locations with respect to the coronary sinus suggested that bipolar shocks, given between two adjacent electrodes on a multipolar catheter, might be more effective for use in left-sided accessory pathway ablation. The canine experiments (17) appeared to confirm this supposition, with a near ideal distribution of fibrosis within the coronary sulcus. Subsequent canine studies using unipolar shocks suggested that the effects would be similar to those from bipolar shocks. Bipolar shocks were used in Patients 1 to 4 and 7 and 8; unipolar shocks were used in Patients 5 and 6. The proportionately larger hearts in human subjects may have prevented sufficient energy from reaching the accessory pathways. Nevertheless, we are concerned about the possibility of coronary artery damage and have adopted a policy of requiring angiography before attempted left-sided accessory pathway ablation. We decided not to recommend an attempted ablation procedure for three patients because coronary angiography demonstrated the presence of a dominant left coronary artery circulation.

Other observations. In the long-term canine studies (17), catheterization and angiography several weeks after the shocks were entirely normal except for coronary sinus occlusion with development of profuse collateral vessels. Asymptomatic coronary sinus occlusion was confirmed in two of our patients 8 to 9 weeks after attempted ablation. Coronary sinus occlusion prevents subsequent attempts at ablation after initial failure. Catheter leads were left in the coronary sinus for electrophysiologic studies on the days after attempted ablation; however, concerns for the integrity of the recently damaged vessel prevented further ablation attempts after the reappearance of accessory pathway conduction. It should be noted that electrode catheters generally are not designed for use in ablation procedures, and many cannot withstand the high energy levels involved (21). Examples of the lead models to be used in any ablation procedure should be tested first in vitro.

Conclusions. Electrical ablation of accessory pathways using the coronary sinus approach was attempted in eight patients, with initially promising but usually transient results. In some patients, several weeks may be required for the development of the full affect, possibly related to a maturing fibrotic process involving the pathway. The technique appears to be safe at energy levels below 100 J. Unfortunately, shocks at these levels have not proved very effective in creating permanent accessory pathway block, and higher levels carry a substantial risk of perforating the coronary sinus. If successful in even a minority of patients, the benefits of a nonsurgical "cure" or a lessened requirement for antiarrhythmic medications might make the procedure useful. Nevertheless, the limited efficacy and the

complications of the technique described in this paper emphasize the experimental nature of the procedure and the need for much further development before widespread application can be contemplated.

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